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=> d l20 1-40 bib ab

L20 ANSWER 1 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2006:1161761 CAPLUS << LOGINID::20080906>>

DN 146:376584

TI Characterization of a new NIH-registered variant human embryonic stem cell line, BG01V: a tool for human embryonic stem cell research

AU Plaia, Todd W.; Josephson, Richard; Liu, Ying; Zeng, Xianmin; Ording, Carol; Toumadje, Arazdordi; Brimble, Sandii N.; Sherrer, Eric S.; Uhl, Elizabeth W.; Freed, William J.; Schulz, Thomas C.; Maitra, Anirban; Rao, Mahendra S.; Auerbach, Jonathan M.

 CS American Type Culture Collection, Stem Cell Center, Manassas, VA, USA

SO Stem Cells (Durham, NC, United States) (2006), 24(3), 531-546 CODEN: STCEEJ; ISSN: 1066-5099

PB AlphaMed Press

DT Journal

LA English

AB Human embryonic stem cells (hESCs) offer a renewable source of a wide range of cell types for use in research and cellbased therapies. Characterizing these cells provides important information about their current state and affords relevant details for subsequent manipulations. For example, identifying genes expressed during culture, as well as their temporal expression order after passaging and conditions influencing the formation of all three germ layers may be helpful for the prodn. of functional beta islet cells used in treating type I diabetes. Although several hESC lines have demonstrated karyotypic instability during extended time in culture, select variant lines exhibit characteristics similar to their normal parental lines. Such variant lines may be excellent tools and abundant sources of cells for pilot studies and in vitro differentiation research in which chromosome no. is not a concern, similar to the role currently played by embryonal carcinoma cell lines. It is crucial that the cells be surveyed at a genetic and proteomic level during extensive propagation, expansion, and manipulation in vitro. Here we describe a comprehensive characterization of the variant hESC line BG01V, which was derived from the karyotypically normal, parental hESC line BG01. Our characterization process employs cytogenetic anal., short tandem repeat and HLA typing, * * * mitochondrial * * * DNA sequencing, * * * gene* * * expression anal. using quant. reverse transcription-polymerase chain reaction and *** microarray***, assessment of telomerase activity, methylation anal., and immunophenotyping

and teratoma formation, in addn. to screening for bacterial, fungal, mycoplasma, and human pathogen contamination.

RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 2 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2006:1145212 CAPLUS << LOGINID::20080906>> DN 146:456152

 $\ensuremath{\mathsf{TI}}$ Chip-based mtDNA mutation screening enables fast and reliable genetic diagnosis of OXPHOS patients

AU Van Eijsden, Rudy G. E.; Gerards, Mike; Eijssen, Lars M. T.; Hendrickx, Alexandra T. M.; Jongbloed, Roselie J. E.; Wokke, John H. J.; Hintzen, Rogier Q.; Rubio-Gozalbo, Maria E.; De Coo, Irenaeus F. M.; Briem, Egill; Tiranti, Valeria; Smeets, Hubert J. M. CS Department of Clinical Genetics, Research Institute Growth and Development, Maastricht University, Maastricht, Neth.

SO Genetics in Medicine (2006), 8(10), 620-627 CODEN: GEMEF3; ISSN: 1098-3600

PB Lippincott Williams & Wilkins

DT Journal

LA English

AB Purpose: Oxidative phosphorylation is under dual genetic control of the nuclear and the mitochondrial DNA (mtDNA). Oxidative phosphorylation disorders are clin. and genetically heterogeneous, which makes it difficult to det. the genetic defect, and symptom-based protocols which link clin. symptoms directly to a specific gene or mtDNA mutation are falling short. Moreover, approx. 25% of the pediatric patients with oxidative phosphorylation disorders is estd. to have mutations in the mtDNA and a std. screening approach for common mutations and deletions will only explain part of these cases. Therefore, we tested a new CHIP-based screening method for the mtDNA. Methods: MitoChip (Affymetrix) resequencing was performed on three test samples and on 28 patient samples. Results: Call rates were 94% on av. and heteroplasmy detection levels varied from 5-50%. A genetic diagnosis can be made in almost one-quarter of the patients at a potential output of 8 complete mtDNA sequences every 4 days. Moreover, a no. of potentially pathogenic unclassified variants (UV) were detected. Conclusions: The availability of long-range PCR protocols and the predominance of single nucleotide substitutions in the mtDNA make the resequencing CHIP a very fast and reliable method to screen the complete mtDNA for mutations.

RE.ONT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 3 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2006:994688 CAPLUS << LOGINID::20080906>> DN 146:331445

TI Detection of mutations in genes associated with hearing loss using a microarray-based approach

AU Siemering, Kirby; Manji, Shehnaaz S. M.; Hutchison, Wendy M.; Du Sart, Desiree; Phelan, Dean; Dahl, Hans-Henrik M. CS. Murdoch Childrens Research Institute, Royal Children's Hospital, University of Melbourne, Parkville, Victoria, Australia SO. Journal of Molecular Diagnostics (2006), 8(4), 483-489 CODEN: JMDIFP; ISSN: 1525-1578

PB American Society for Investigative Pathology and the Association for Molecular Pathology

DT Journal

LA English

AB Knowing the etiol. of hearing loss in a person has implications for counseling and management of the condition. More than 50% of cases of early onset, nonsyndromic

sensorineural hearing loss are attributable to genetic factors. However, deafness is a genetically heterogeneous condition and it is therefore currently not economically and practically feasible to screen for mutations in all known deafness genes. The authors have developed a microarray-based hybridization biochip assay for the detection of known mutations. The current version of the hearing loss biochip detects nine common mutations in the connexin 26 gene, four mutations in the pendrin gene, one mutation in the usherin gene, and one mutation in mitochondrial DNA. The biochip was validated using DNA from 250 people with apparent nonsyndromic, moderate to profound sensorineural hearing loss. The hearing loss biochip detected with 100% accuracy the mutations it was designed for. No false-positives or false-neg, results were seen. The biochip can easily be expanded to test for addnl. mutations in genes assocd. with hearing impairment or other genetic conditions.

RE.ONT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 4 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2006:953134 CAPLUS < LOGINID::20080906>>

DN 146:182317

TI Metallothioneins and zinc dysregulation contribute to neurodevelopmental damage in a model of perinatal viral infection

AU Williams, Brent L.; Yaddanapudi, Kavitha; Kirk, Cassandra M.; Soman, Arya; Hornig, Mady; Lipkin, W. Ian

CS Greene Infectious Disease Laboratory, Mailman School of Public Health, Columbia University, New York, NY, USA

SO Brain Pathology (2006), 16(1), 1-14 CODEN: BRPAE7; ISSN: 1015-6305

PB Blackwell Publishing Ltd.

DT Journal

LA English

AB Neonatal Borna disease (NBD) virus infection in the Lewis rat results in life-long viral persistence and causes behavioral and neurodevelopmental abnormalities. A hallmark of the disorder is progressive loss of cerebellar Purkinje and dentate gyrus granule cells. Findings of increased brain metallothionein-I and -II (***MT*** -I/-II) mRNA expression in ***cDNA** *** microarray* * * expts. led us to investigate MT isoforms and their relationship to brain zinc metab., cellular toxicity, and neurodevelopmental abnormalities in this model. Real-time PCR confirmed marked induction of MT-I/-II mRNA expression in the brains of NBD rats (40.5-fold increase in cerebellum, p<0.0001; 6.8-fold increase in hippocampus, p = 0.003; and 9.5-fold increase in striatum, p = 0.0012), whereas a trend toward decreased MT-III mRNA was found in hippocampus (1.25-fold decrease, p = 0.0841). Double label immunofluorescence revealed prominent MT-I/-II expression in astrocytes throughout the brain; MT-III protein was decreased in granule cell neurons and increased in astrocytes, with differential subcellular distribution from cytoplasmic to nuclear compartments in NBD rat hippocampus. Modified Timm staining of hippocampus revealed reduced zinc in mossy fiber projections to the hilus and CA3, accumulation of zinc in glial cells and degenerating granule cell somata, and robust mossy fiber sprouting into the inner mol. layer of the dentate gyrus. Zinc Transporter 3 (ZnT-3) mRNA expression was decreased in hippocampus (2.3-fold decrease, p = 0.0065); staining for its correlate protein was reduced in hippocampal mossy fibers. Furthermore, 2 mols. implicated in axonal pathfinding and mossy fiber sprouting, the extracellular matrix glycoprotein, tenascin-R (TN-R), and the hyaluronan receptor CD44, were increased in NBD hippocampal neuropil. Abnormal zinc metab. and mechanisms of neuroplasticity may

contribute to the pathogenesis of disease in this model, raising more general implications for neurodevelopmental damage following viral infections in early life.

RE.ONT 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 5 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2006:941880 CAPLUS << LOGINID::20080906>> DN 146:1136

TI Quantitation of heteroplasmy of mtDNA sequence variants identified in a population of AD patients and controls by array-based resequencing

AU Coon, Keith D.; Valla, Jon; Szelinger, Szabolics; Schneider, Lonnie E.; Niedzielko, Tracy L.; Brown, Kevin M.; Pearson, John V.; Halperin, Rebecca; Dunckley, Travis; Papassotiropoulos, Andreas; Caselli, Richard J.; Reiman, Eric M.; Stephan, Dietrich A. CS Neurogenomics Division, Translational Genomics Research Institute, Phoenix, AZ, 85004, USA

SO Mitochondrion (2006), 6(4), 194-210 CODEN: MITOCN; ISSN: 1567-7249

PB Elsevier B.V.

DT Journal

LA English

AB The role of mitochondrial dysfunction in the pathogenesis of Alzheimer's disease (AD) has been well documented. Though evidence for the role of mitochondria in AD seems incontrovertible, the impact of mitochondrial DNA (mtDNA) mutations in AD etiol. remains controversial. Though mutations in mitochondrial-encoded genes have repeatedly been implicated in the pathogenesis of AD, many of these studies have been plagued by lack of replication as well as potential contamination of nuclear-encoded mitochondrial pseudogenes. To assess the role of mtDNA mutations in the pathogenesis of AD, while avoiding the pitfalls of nuclear-encoded mitochondrial pseudogenes encountered in previous investigations and showcasing the benefits of a novel resequencing technol., we sequenced the entire coding region (15,452 bp) of mtDNA from 19 extremely well-characterized AD patients and 18 agematched, unaffected controls utilizing a new, reliable, highthroughput array-based resequencing technique, the Human MitoChip. High-throughput, ***array*** -based DNA resequencing of the entire ***mtDNA*** coding region from platelets of 37 subjects revealed the presence of 208 loci displaying a total of 917 sequence variants. There were no statistically significant differences in overall mutational burden between cases and controls, however, 265 independent sites of statistically significant change between cases and controls were identified. Changed sites were found in genes assocd with complexes I (30.2%), III (3.0%), IV (33.2%), and V (9.1%) as well as tRNA (10.6%) and rRNA (14.0%). Despite their statistical significance, the subtle nature of the obsd. changes makes it difficult to det. whether they represent true functional variants involved in AD etiol. or merely naturally occurring dissimilarity. Regardless, this study demonstrates the tremendous value of this novel mtDNA resequencing platform, which avoids the pitfalls of erroneously amplifying nuclear-encoded mtDNA pseudogenes, and our proposed anal. paradigm, which utilizes the availability of raw signal intensity values for each of the four potential alleles to facilitate quant. ests. of mtDNA heteroplasmy. This information provides a potential new target for burgeoning diagnostics and therapeutics that could truly assist those suffering from this devastating disorder.

RE.ONT 79 THERE ARE 79 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 6 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2006:722407 CAPLUS << LOGINI D:: 20080906>>

DN 145:487091

TI Molecular analysis of ANT1, TWI NKLE and POLG in patients with multiple deletions or depletion of mitochondrial DNA by a dHPLC-based assay

AU Naimi, Mourad; Bannwarth, Sylvie; Procaccio, Vincent; Pouget, Jean; Desnuelle, Claude; Pellissier, Jean-Francois; Roetig, Agnes; Munnich, Arnold; Calvas, Patrick; Richelme, Christian; Jonveaux, Philippe; Castelnovo, Giovanni; Simon, Melvin; Clanet, Michel; Wallace, Douglas; Paquis-Flucklinger, Veronique

CS Department of Medical Genetics, Archet 2 Hospital, CHU, Nice, Fr.

SO European Journal of Human Genetics (2006), 14(8), 917-922 CODEN: EJHGEU; ISSN: 1018-4813

PB Nature Publishing Group

DT Journal

LA English

AB ANT1, TWINKLE and POLG genes affect mtDNA stability and are involved in autosomal dominant PEO, while mutations in POLG are responsible for numerous clin. presentations, including autosomal recessive PEO, sensory ataxic neuropathy, dysarthria and ophthalmoparesis (SANDO), spino-cerebellar ataxia and epilepsy (SCAE) or Alpers syndrome. In this study, we report on the mutational anal. of ANT1, TWINKLE and POLG genes in 15 unrelated patients, using a dHPLC-based protocol. This series of patients illustrates the large ***array*** of clin. presentations assocd. with ***mtDNA*** stability defects, ranging from isolated benign PEO to fatal Alpers syndrome. A total of seven different mutations were identified in six of 15 patients (40%). Six different recessive mutations were found in POLG, one in TWI NKLE while no mutation was identified in ANT1. Among the POLG mutations, three are novel and include two missense and one frameshift changes. Seventeen neutral changes and polymorphisms were also identified, including four novel neutral polymorphisms. Overall, this study illustrates the variability of phenotypes assocd. with mtDNA stability defects, increases the mutational spectrum of POLG variants and provides an efficient and reliable detection protocol for ANT1, TWINKLE and POLG mutational screening.

RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 7 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2006:689698 CAPLUS < < LOGINID::20080906>>

DN 145:370272

TI Genesis and wanderings: origins and migrations in asymmetrically replicating mitochondrial DNA

AU Brown, Timothy A.; Clayton, David A.

CS Howard Hughes Medical Institute, Ashburn, VA, USA

SO Cell Cycle (2006), 5(9), 917-921 CODEN: CCEYAS; ISSN: 1538-4101

PB Landes Bioscience

DT Journal; General Review

LA English

AB A review and discussion. Mammalian mitochondria maintain a small circular genome that encodes RNA and polypeptides that are essential for the generation of ATP through oxidative phosphorylation. The mechanism of replication of mammalian mitochondrial DNA (mtDNA) has recently been a topic of controversy. New evidence has led to a modified strand-displacement model that reconciles much of the current data. This revision stems from a new appreciation for alternative light-

strand origins. The authors consider here some of the potential mechanisms for light-strand origin initiation. They also consider further the susceptibility of branch migration within replicating mtDNA mols. The existence of alternative light-strand origins and a propensity for branch migration in replicating *** mtDNA*** mols. exposes a new *** array*** of possible configurations of *** mtDNA***. The assortment and assignment of these forms is relevant to the interpretation of exptl. data and may also yield insight into the mol. basis of replication errors.

RE ONT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 8 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2006:385086 CAPLUS < < LOGINID::20080906>>

DN 144:485663

TI Cardiac genomic response following preconditioning stimulus

AU Das, Dipak K.; Maulik, Nilanjana

CS Cardiovascular Research Center, University of Connecticut School of Medicine, Farmington, CT, 06030-1110, USA

SO Cardiovascular Research (2006), 70(2), 254-263 CODEN: CVREAU; ISSN: 0008-6363

PB Elsevier B.V.

DT Journal; General Review

LA English

AB A review. This review focuses on the genomic response following a preconditioning stimulus. Initial studies demonstrated that classical ischemic preconditioning mediated by cyclic episodes of short durations of reversible ischemia and reperfusion could result in the reprogramming of gene expression. Some of these genes are translated into proteins during the late preconditioning or so-called "second window of protection". Subsequent studies detd. a unique similarity of the expressed gene profiles between diverse varieties of preconditioning including ischemic/hypoxic, heat shock, and oxidative stress. The most common genes that are expressed by virtually any kind of stress conditioning include antioxidants like superoxide dismutase, glutathione peroxidase and heme oxygenase and heat shock proteins such as HSP70. At a later date, differential display and subtractive hybridization techniques revealed the identities of many other genes including those belonging to mitochondrial respiratory chain such as ATPases. More recently, gene ***array*** profiles using gene chips detd. several other triggered by preconditioning including the *** mitochondrial*** genes. The results of the studies present in the literature clearly indicate the existence of a strong resemblance between the patterns of gene expression profiles induced by diverse preconditioning stimuli, oxidative stress being situated at the cross-roads of all forms of the stresses. Redox signaling appears to be responsible for the conversion of the ischemia/reperfusion- induced "death signal" into preconditioning-mediated "survival signal". RE. CNT 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE **FORMAT**

L20 ANSWER 9 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2005:228660 CAPLUS << LOGINID::20080906>> DN 142:458434

TI Dose-dependent transcriptome changes by metal ores on a human acute lymphoblastic leukemia cell line AU Sun, Nina N.; Fastje, Cynthia D.; Wong, Simon S.; Sheppard, Paul R.; MacDonald, Stephanie J.; Ridenour, Gary;

Hyde, Juanita D.; Witten, Mark L.

CS Southwest Environmental Science Center and Department of Pediatrics, University of Arizona College of Medicine, Tucson, USA SO Toxicology and Industrial Health (2003), 19(7-10), 157-163 CODEN: TIHEEC: ISSN: 0748-2337

PB Arnold, Hodder Headline

DT Journal

LA English

AB The increased morbidity of childhood leukemia in Fallon, Nevada and Sierra Vista, Arizona has prompted great health concern. The main characteristic that these two towns share is the environmental pollution attributed to metal ore from abandoned mining operations. Consequently, the authors have investigated the transcriptome effects of metal ores from these endemic areas using a human T-cell acute lymphoblastic leukemia cell line (T-ALL). Metal ore from Fallon significantly increased cell growth after 24, 48 and 72 h of incubation at 1.5 .mu.g/mL concn., as measured by trypan-blue. Sierra Vista ore significantly increased cell growth with 0.15 and 1.5 .mu.g/mL following 72 h of incubation. From human cDNA

microarray*, results indicate that in total, eight

genes , mostly metallothionein (***MT***)

genes , were up-regulated and 10 genes were downregulated following treatment of the T-ALL cells with 0.15 and 1.5 .mu.g/mL of metal ores at 72 h, in comparison with untreated cells. Twenty-eight metals of both ores were quantified and their presence may be assocd. with the cell growth rate and dosedependent activation of transcriptomes in immature T-cells. RE ONT 25 THERE ARE 25 CITED REFERENCES AVAILABLE ALL CITATIONS AVAILABLE IN THE RE FOR THIS RECORD **FORMAT**

L20 ANSWER 10 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2003:766755 CAPLUS << LOGINID::20080906>> DN 140:13509

TI Functionalized self-assembled monolayer on gold for detection of human mitochondrial tRNA gene mutations AU Du, Weidong; Marsac, Cecile; Kruschina, Margit; Ortigao, Havio; Horentz, Catherine

CS Interactiva Division, ThermoHybaid, Ulm, 89077, Germany SO Analytical Biochemistry (2003), 322(1), 14-25 CODEN: ANBCA2; ISSN: 0003-2697

PB Elsevier Science

DT Journal

LA English

AB We developed a rapid and simple method to identify singlenucleotide polymorphisms (SNPs) in the human mitochondrial tRNA genes. This method is based on a universal, functionalized, self-assembled monolayer, XNA on Gold chip platform. A set of probes sharing a given allele-specific sequence with a single base substitution near the middle of the sequence was immobilized on chips and the chips were then hybridized with fluorescencelabeled ref. targets produced by asym. polymerase chain reaction from patient DNA. The ratio of the hybridization signals from the ref. and test targets with each probe was then calcd. A ratio of above 3 indicates the presence of a wild-type sequence and a ratio of below 0.3 indicates a mutant sequence. We tested the sensitivity of the chip for known mutations in tRNALeu(UUR) and tRNALys genes and found that it can also be used to discriminate multiple mutations and heteroplasmy, two typical features of human mitochondrial DNA. The XNA on Gold biochip method is a simple and rapid microarray method that can be used to test rapidly and reliably any SNP in the mitochondrial genome or elsewhere. It will be particularly useful for detecting SNPs assocd. with human diseases.

RE.ONT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE **FORMAT**

L20 ANSWER 11 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2003:592105 CAPLUS << LOGINI D:: 20080906>> DN 139:228515

TI Hepatic gene expression in histologically progressive nonalcoholic steatohepatitis

AU Sreekumar, Raghavakaimal; Rosado, Barbara; Rasmussen, Deborah; Charlton, Michael

CS Division of Gastroenterology and Hepatology, Mayo Foundation, Rochester, MN, USA

SO Hepatology (Philadelphia, PA, United States) (2003), 38(1), 244-251 CODEN: HPTLD9; ISSN: 0270-9139

PB W. B. Saunders Co.

DT Journal

LA English

AB Although the mol. basis for the pathophysiol. of nonalcoholic steatohepatitis (NASH) is poorly understood, insulin resistance and mitochondrial dysfunction are physiol. hallmarks of this condition. We sought evidence of a transcriptional or pretranscriptional basis for insulin resistance and

*** mitochondrial*** dysfunction through measurement of hepatic *** gene*** expression (mRNA) using high-d. synthetic oligonucleotide ***microarray*** anal. (Hu6800 GeneChip, Affymetrix, CA). Global hepatic gene expression was detd. in snap-frozen liver biopsy specimens from 4 groups: (1) patients with cirrhotic-stage NASH (n = 6), (2) patients with cirrhosis caused by hepatitis C virus (HCV) (n = 6), (3) patients with cirrhosis secondary to primary biliary cirrhosis (PBC) (n = 6), and (4) healthy controls (n = 6). Genes were considered to be expressed differentially in NASH only if there was a greater than 2-fold difference in abundance of mRNA when compared with each of the control groups. Sixteen genes were uniquely differentially expressed (4 overexpressed and 12 underexpressed) in patients with cirrhotic-stage NASH. Genes that were significantly underexpressed included genes important for maintaining mitochondrial function (copper/zinc superoxide dismutase, aldehyde oxidase, and catalase). Glucose 6phosphatase, alc. dehydrogenase, elongation factor-TU, methylglutaryl CoA (CoA), acyl CoA synthetase, oxoacyl CoA thiolase, and ubiquitin also were underexpressed in NASH. Genes that were overexpressed in NASH included complement component C3 and hepatocyte-derived fibringen-related protein. potentially contributing to impaired insulin sensitivity. In conclusion, these studies provide evidence for a transcriptional or pretranscriptional basis for impaired mitochondrial function (attenuated capacity for the dismutation of reactive oxygen species) and diminished insulin sensitivity (increased acute phase reactants) in patients with histol. progressive NASH. Further studies are required to det. the mechanism and the physiol. significance of these findings.

RE. CNT 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE **FORMAT**

L20 ANSWER 12 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2002:969223 CAPLUS << LOGINID::20080906>> DN 138:216353

TI Heteroplasmy and evidence for recombination in the mitochondrial control region of the flatfish Platichthys flesus AU Hoarau, Galice; Holla, Suzanne; Lescasse, Rachel; Stam, Wytze T.; Olsen, Jeanine L.

CS Department of Marine Biology, Centre for Ecological and Evolutionary Studies, University of Groningen, Haren, 9750 AA, Neth.

SO Molecular Biology and Evolution (2002), 19(12), 2261-2264 CODEN: MBEVEO; ISSN: 0737-4038

PB Society for Molecular Biology and Evolution

DT Journal

LA English

AB The general assumption that mitochondrial DNA (mtDNA) does not undergo recombination has been challenged recently in invertebrates. Here the authors present the first direct evidence for recombination in the mtDNA of a vertebrate, the flounder Platichthys flesus. The control region in the mtDNA of this flatfish is characterized by the presence of a variable no. of tandem repeats and a high level of heteroplasmy. Two types of repeats were recognized, differing by two C-T point mutations. Most individuals carry a pure "C" or a pure "T" array, but one individual showed a compd. "CT" array. Such a compd.

array is evidence for recombination in the

*** mtDNA*** control region from the flounder.

RE.ONT 22 THERE ARE 22 CITED REFERENCES AVAILABLE

FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 13 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2002:841725 CAPLUS < LOGINID::20080906>>

DN 138:216104

TI Evolution of heteroplasmy at a mitochondrial tandem repeat locus in cultured rabbit cells

AU Casane, Didier; Gueride, Monique

CS Batiment Fermat, Laboratoire de Biologie Cellulaire, Universite de Versailles Saint-Quentin-en-Yvelines, Versailles, 78035, Fr.

SO Current Genetics (2002), 42(1), 66-72 CODEN: CUGED5; ISSN: 0172-8083

PB Springer-Verlag

DT Journal

LA English

AB Surveys of animal mitochondrial DNA (mtDNA) polymorphism reveal that mtDNA length variation is common. Much of this variation involves non-coding tandem repeat arrays in the main control region of the mol. Despite a high mutation rate, generating systematic individual ***mtDNA*** length heteroplasmy, the no. of repeats in a tandem ***array*** is maintained within a narrow range in lagomorphs. To investigate the basis for this apparent paradox, we studied the evolution of mtDNA length polymorphism in several rabbit cell clones contg. different proportions of mtDNA, with four or five 153-bp repeats. Our data show that equiv. amts. of two mtDNA mol. types are not stable (evolution towards a predominant type being the rule) and that other types remain represented, maintaining the length polymorphism. The data suggest that ***mtDNA*** with a longer ***array*** of repeats have a replicative advantage that could depend on the nuclear background. RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE **FORMAT**

L20 ANSWER 14 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2002:72748 CAPLUS < < LOGINI D::20080906>>

DN 136:146104

TI Human stress genes identified using DNA microarrays

IN Chenchik, Alex; Lukashev, Matvey E.

PA Clontech Laboratories, Inc., USA

SO U.S. Pat. Appl. Publ., 57 pp., Cont.-in-part of U.S. Ser. No. 441,920. CODEN: USXXCO

DT Patent

LA English

PI US 20020009730 A1 20020124 US 2001-782909 20010213

PRAI US 1998-222256 B2 19981228 US 1999-440305 B2 19991117 US 1999-441920 A2 19991117

AB Human stress gene arrays and methods for their use are provided. The subject arrays include a plurality of polynucleotide spots, each of which is made up of a polynucleotide probe compn. of unique polynucleotides corresponding to a human stress gene. The av. length of the polynucleotide probes is 50-1000 nucleotides. The d. of the spots on the array did not exceed 400/cm2 and the spots had a diam. ranging between 10 and 5000 .mu.m. Furthermore, the no. of polynucleotide probe spots on the array ranged between 50 and 2000 nucleotides. The subject arrays find use in hybridization assays, particularly in assays for the identification of differential gene expression of human stress genes. Two hundred thirty-six different human stress genes were identified using this approach.

L20 ANSWER 15 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2002:16642 CAPLUS << LOGINID::20080906>>

DN 136:399728

TI Blood genomic responses differ after stroke, seizures, hypoglycemia, and hypoxia: blood genomic fingerprints of disease

AU Tang, Yang; Lu, Aigang; Aronow, Bruce J.; Sharp, Frank R. CS Department of Neurology and Neuroscience Program, University of Cincinnati, Cincinnati, OH, 45267-0536, USA SO Annals of Neurology (2001), 50(6), 699-707 CODEN: ANNED3: ISSN: 0364-5134

PB Wiley-Liss, Inc.

DT Journal

LA English

AB Using microarray technol., the authors investigated whether the gene expression profile in white blood cells could be used as a fingerprint of different disease states. Adult rats were subjected to ischemic strokes, hemorrhagic strokes, sham surgeries, kainate-induced seizures, hypoxia, or insulin-induced hypoglycemia, and compared with controls. The white blood cell RNA expression patterns were assessed 24 h later using oligonucleotide microarrays. Results showed that many genes were upregulated or downregulated at least 2-fold in white blood cells after each exptl. condition. Blood genomic response patterns were different for each condition. These results demonstrate the potential of blood gene expression profiling for diagnostic, mechanistic, and therapeutic assessment of a wide variety of disease states.

RE.ONT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 16 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2001:888406 CAPLUS << LOGI NI D:: 20080906>> DN 136:183012

TI Modulation of intestinal gene expression by dietary zinc status: effectiveness of cDNA arrays for expression profiling of a single nutrient deficiency

AU Blanchard, Raymond K.; Moore, J. Bernadette; Green, Calvert L.; Cousins, Robert J.

CS Food Science and Human Nutrition Department and Center for Nutritional Sciences, University of Florida, Gainesville, FL, 32611-0370, USA

SO Proceedings of the National Academy of Sciences of the United States of America (2001), 98(24), 13507-13513 CODEN: PNASA6; ISSN: 0027-8424

PB National Academy of Sciences

DT Journal

LA English

AB The authors tested the feasibility of using cDNA arrays to compare the global changes in expression of genes of known function that occur in the early stages of rodent zinc deficiency. The gene-modulating effects of this deficiency were demonstrated by real-time quant. PCR measurements of altered mRNA levels for metallothionein 1, zinc transporter 2, and uroquanylin, all of which have been previously documented as zinc-regulated genes. As a result of the low level of inherent noise within this model system and application of a recently reported statistical tool for statistical anal. of microarrays, the authors demonstrate the ability to reproducibly identify the modest changes in mRNA abundance produced by this single micronutrient deficiency. Among the genes identified by this array profile are intestinal genes that influence signaling pathways, growth, transcription, redox, and energy utilization. Addnl., the influence of dietary zinc supply on the expression of some of these genes was confirmed by real-time quant. PCR. Overall, these data support the effectiveness of cDNA array expression profiling to investigate the pleiotropic effects of specific nutrients and may provide an approach to establishing markers for assessment of nutritional status. RE.ONT 27 THERE ARE 27 CITED REFERENCES AVAILABLE

L20 ANSWER 17 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2001:879728 CAPLUS < LOGINI D::20080906>> DN 136:148992

ALL CITATIONS AVAILABLE IN THE RE

TI Gene expression differences in bipolar disorder revealed by cDNA array analysis of post-mortem frontal cortex

AU Bezchlibnyk, Yarema B.; Wang, Jun-Feng; McQueen, Glenda M.; Young, L. Trevor

CS Department of Psychiatry and Behavioural Neurosciences, McMaster University, Hamilton, ON, L8N 3Z5, Can.

SO Journal of Neurochemistry (2001), 79(4), 826-834 CODEN: JONRA9: ISSN: 0022-3042

PB Blackwell Science Ltd.

FOR THIS RECORD

FORMAT

DT Journal

LA English

AB Previous studies have implicated a no. of biochem. pathways in the etiol. of bipolar disorder (BD). However, the precise abnormalities underlying this disorder remain to be established. To investigate novel factors that may be important in the pathophysiol, of BD, we utilized cDNA expression arrays to examine differences in expression of up to 1200 genes known to be involved in potentially relevant biochem. processes. This investigation was undertaken in post-mortem samples of frontal cortex tissue from patients with BD and matched controls, obtained (n = 10/group) from the Stanley Foundation Neuropathol. Consortium. Results include significant (greater than 35% change in signal intensity) differences between BD and controls in a no. of genes (n = 24). Selected targets were analyzed by RT-PCR, which confirmed a decrease in transforming growth factor-beta 1 (TGF-.beta.1), and an increase in both caspase-8 precursor (casp-8) and transducer of erbB2 (Tob) expression in BD. We further obsd. a significant decrease of TGF-.beta.1 mRNA levels in BD by RT-PCR in individual postmortem samples. Given the neuroprotective role attributed to this inhibitory cytokine, our results suggest that the downregulation of TGF-.beta.1 may lead to various neurotoxic insults potentially involved in the etiol. of certain mood disorders.

RE.CNT 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 18 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2001:750592 CAPLUS << LOGINID::20080906>> DN 136:277222

TI Identification of candidate genes in ulcerative colitis and Crohn's disease using cDNA array technology

AU Uthoff, Sonja M. S.; Eichenberger, M. Robert; Lewis, Robert K.; Fox, Matthew P.; Hamilton, Crystal J.; Mcauliffe, Tracy L.; Grimes, H. Leighton; Galandiuk, Susan

CS Digestive Surgery Research Laboratory, Department of Surgery, University of Louisville School of Medicine, Louisville, KY, USA

SO International Journal of Oncology (2001), 19(4), 803-810 CODEN: IJONES; ISSN: 1019-6439

PB International Journal of Oncology

DT Journal

LA English

AB Inflammatory bowel disease (IBD) follows a multigenic mode of inheritance, encompassing the clin. discrete phenotypes of ulcerative colitis (UC) and Orohn's disease (CD). The risk of malignant transformation of the colon increases with the duration and extent of IBD and is particularly high for patients with a longstanding history of UC. We wished to identify candidate genes that might be involved in disease pathogenesis based on functional plausibility and their putative role in IBD carcinogenesis. PolyA+ mRNA prepn. from inflamed intestinal mucosa of patients with a longstanding history of UC and CD was performed with subsequent hybridization of a phosphorus [.alpha.-32P]-dATP-labeled cDNA populations to nucleic acid arrays. Of 588 different human gene transcripts arrayed, secreted apoptosis-related protein 1 (Sarp1), frizzled (fz) homologs, and disheveled (dvl) were differentially expressed, being elevated in UC as compared to CD. These genes encode proteins involved in the Wingless-type (Wnt)/ss-catenin signaling pathway. The autonomous expression of Sarp1 and Sarp1compatible fz receptor genes suggests that the Wnt pathway may be involved in UC carcinogenesis.

RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 19 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2001:427721 CAPLUS << LOGINI D::20080906>> DN 136:35504

TI Expression profiling of gastric adenocarcinoma using cDNA array

AU El-Rifai, Wa'el; Frierson, Henry F., Jr.; Harper, Jeffrey C.; Powell, Steven M.; Knuutila, Sakari

CS Department of Medicine, University of Virginia Health Systems, Charlottesville, VA, 22908-0708, USA

SO International Journal of Cancer (2001), 92(6), 832-838 CODEN: IJCNAW; ISSN: 0020-7136

PB Wiley-Liss, Inc.

DT Journal

LA English

AB To investigate the expression profile of gastric adenocarcinoma, cDNA array expts. were performed using Atlas Human Cancer 1.2 K Array (Clontech Labs., Palo Alto, CA) on nine xenografted and two primary gastric cancer samples. The expression of the tumor samples was compared to that of two normal gastric epithelial tissues. The expression pattern of the

primary tumors was similar to that of xenografted tumors. The up-regulated genes had expression ratios ranging from 2.5 to 16, whereas the down-regulated genes had a range from -2.5 to -16. No variation in gene expression was detected in the anal. of the xenografted tumors vs. the primary tumors, indicating that the xenografts represented the primary tumors well. Thirty-eight genes showed altered gene expression in 5 or more samples (>45%). Thirty-one genes were up-regulated and seven genes were down-regulated. The most abundantly up-regulated genes (ratio > 5) included genes such as S100A4, CDK4, MMP14 and beta catenin. The GIF was markedly down-regulated (ratio < 10). To confirm our findings, six genes (three up- and three down-regulated) were selected for semiguant. RT-PCR anal. The RT-PCR results were consistent with the array findings. Our approach revealed that several genes are abnormally expressed in gastric cancer and found that genes known to interact in several common mol. pathway(s) were consistently altered. RE ONT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE **FORMAT**

L20 ANSWER 20 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2001:312014 CAPLUS << LOGINID::20080906>> DN 136:64938

TI Toward elucidating the global gene expression patterns of developing Arabidopsis: parallel analysis of 8 300 genes by a high-density oligonucleotide probe array

AU Zhu, Tong; Budworth, Paul; Han, Bin; Brown, Devon; Chang, Hur-Song; Zou, Guangzhou; Wang, Xun CS Torrey Mesa Research Institute, Inc., San Diego, CA, 92121,

USA SO Plant Physiology and Biochemistry (Paris, France) (2001), 39(3-4), 221-242 CODEN: PPBLEX: ISSN: 0981-9428

PB Editions Scientifiques et Medicales Elsevier

DT Journal

LA English

FOR THIS RECORD

DN 134:16185

FORMAT

Arabidopsis thaliana has been widely used as a model system, in various aspects of biol. studies, such as genomics, genetics, cellular, developmental and mol. biol. In order to reveal the mol. events and regulatory networks controlling Arabidopsis development and responses to genetic and environmental changes, we designed and used a high-d. oligonucleotide probe array (GeneChip) to profile global gene expression patterns. The Arabidopsis oligonucleotide probe array consists of probes from 8 300 unique Arabidopsis genes, which covers approx. one-third of the genome. Global transcription profiles of A. thaliana in various developmental stages, and their responses to different environments were generated using this microarray, and archived. Here, we analyze data sets derived from nineteen independent expts. Constitutively and differentially expressed genes in seedlings, roots, leaves, inflorescences, flowers and siliques at different developmental stages were identified. Functions of these genes based on homologs were detd. and categorized. Our results provide insight into the coordinated transcriptional regulation of the genes during plant growth and development. RE.ONT 43 THERE ARE 43 CITED REFERENCES AVAILABLE

L20 ANSWER 21 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2000:701330 CAPLUS << LOGINI D::20080906>>

ALL CITATIONS AVAILABLE IN THE RE

TI Large-scale mitochondrial DNA deletions in skeletal muscle of patients with end-stage renal disease

AU Lim, P.-S.; Cheng, Y.-M.; Wei, Y.-H.

CS Department of Biochemistry and Center for Cellular and Molecular Biology, National Yang-Ming University, Taipei, Taiwan SO Free Radical Biology & Medicine (2000), 29(5), 454-463 CODEN: FRBMEH: ISSN: 0891-5849

PB Elsevier Science Inc.

DT Journal

LA English

AB End-stage renal disease (ESRD) is assocd. with enhanced oxidative stress. This disease state provides a unique system for investigating the deleterious effect of exogenous sources of free radicals and reactive oxygen species (ROS) on mitochondrial DNA (mtDNA). To test the hypothesis that uremic milieu might cause more severe damage to mtDNA, we investigated the prevalence and abundance of mtDNA deletions in the skeletal muscles of ESRD patients. The results showed that the frequencies of occurrence of the 4977 bp and 7436 bp deletions of mtDNA in the muscle tissues of the older ESRD patients were higher than those of the younger patients. The frequency of occurrence of the 4977 bp-deleted mtDNA in the muscle was 33.3% for the patients in the age group of <40 yr, 66.6% in the 41-60-yr-old group, 100% in the 61-80-yr-old group, and 100% in patients >80 yr of age, resp. Only 22% of the normal aged controls carried the 4977 bp mtDNA deletion, whereas 77% (17/22) of the ESRD patients exhibited the mtDNA deletion. Using a semiguant. PCR method, we detd. the proportion of the 4977 bpdeleted mtDNA from the muscles that had been confirmed to harbor the deletion. We found that the proportions of the 4977 bp-deleted mtDNA in the muscle were significantly higher than those of the aged matched controls. Using long-range PCR techniques, a distinctive ***array*** of ***mtDNA** deletions was demonstrated in the muscle of uremic patients. In summary, we found diverse and multiple mtDNA deletions in the skeletal muscles of ESRD patients. These deletions are more prevalent and abundant in ESRD patients than those found in normal populations. Accumulation of uremic toxins and impaired free radical scavenging systems may be responsible for the increased oxidative stress in ESRD patients. Such stress may result in oxidative damage and aging-assocd. mutation of the mitochondrial genome.

RE.ONT 50 THERE ARE 50 CITED REFERENCES AVAILABLE ALL CITATIONS AVAILABLE IN THE RE FOR THIS RECORD **FORMAT**

L20 ANSWER 22 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 2000:614795 CAPLUS << LOGINID::20080906>> DN 133:294551

TI Host gene regulation during coxsackievirus B3 infection in mice: assessment by microarrays

AU Taylor, Lydia A.; Carthy, Christopher M.; Yang, Decheng; Saad, Kareem; Wong, Donald; Schreiner, George; Stanton, Lawrence W.; McManus, Bruce M.

CS Cardiovascular Research Laboratory, Department of Pathology and Laboratory Medicine, St. Paul's Hospital, University of British Columbia, Vancouver, BC, V6Z 1Y6, Can.

SO Circulation Research (2000), 87(4), 328-334 CODEN: CIRUAL; ISSN: 0009-7330

PB Lippincott Williams & Wilkins

DT Journal

LA English

AB Host genetic responses that characterize enteroviral myocarditis have not yet been detd. The injurious and inflammatory process in heart muscle may reflect host responses of benefit to the virus and ultimately result in congestive heart failure and dilated cardiomyopathy. On the other hand, host responses within the myocardium may secure the host against

acute or protracted damage. To investigate the nature of

modified gene expression in comparison with normal tissue, mRNA species were assessed in myocardium using cDNA microarray technol. at days 3, 9, and 30 after infection. Of 7000 clones initially screened, 169 known genes had a level of expression significantly different at 1 or more postinfection time points as compared with baseline. The known regulated genes were sorted according to their functional groups and normalized expression patterns and, subsequently, interpreted in the context of viremic, inflammatory, and healing phases of the myocarditic process.

RE.ONT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 23 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2000:521379 CAPLUS < LOGINID::20080906>>

DN 133:217973

TI Thyroid hormone regulation of hepatic genes in Vivo detected by complementary DNA microarray

AU Feng, Xu; Jiang, Yuan; Meltzer, Paul; Yen, Paul M.
CS Molecular Regulation and Neuroendocrinology Section
Clinical Endocrinology Branch, National Institute of Diabetes and
Digestive and Kidney Diseases, National Institutes of Health,
Bethesda, MD, 20892, USA

SO Molecular Endocrinology (2000), 14(7), 947-955 CODEN: MOENEN; ISSN: 0888-8809

PB Endocrine Society

DT Journal

LA English

AB The liver is an important target organ of thyroid hormone. However, only a limited no. of hepatic target genes have been identified, and little is known about the pattern of their regulation by thyroid hormone. We used a quant. fluorescent cDNA microarray to identify novel hepatic genes regulated by thyroid hormone. Fluorescent-labeled cDNA prepd. from hepatic RNA of T3-treated and hypothyroid mice was hybridized to a cDNA microarray, representing 2225 different mouse genes, followed by computer anal. to compare relative changes in gene expression. Fifty five genes, 45 not previously known to be thyroid hormone-responsive genes, were found to be regulated by thyroid hormone. Among them, 14 were pos. regulated by thyroid hormone, and unexpectedly, 41 were neg. regulated. The expression of 8 of these genes was confirmed by Northern blot analyses. Thyroid hormone affected gene expression for a diverse range of cellular pathways and functions, including gluconeogenesis, lipogenesis, insulin signaling, adenylate cyclase signaling, cell proliferation, and apoptosis. This is the first application of the microarray technique to study hormonal regulation of gene expression in vivo and should prove to be a powerful tool for future studies of hormone and drug action. RE.ONT 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE **FORMAT**

L20 ANSWER 24 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2000:496055 CAPLUS << LOGINID::20080906>>

DN 133:247738

TI DNA chips for fast use

AU Lange, Heidrun

CS Germany

SO LaborPraxis (2000), 24(1), 22-24 CODEN: LAPRDE; ISSN: 0344-1733

PB Vogel Verlag und Druck

DT Journal

LA German

AB The complete cDNA microarray system Micromax allowing comparing gene expression analyses was described. Low sample amts. of 2-4 .mu.g RNA and the detection of lowly exprimed mRNA were possible by the tyramid signal amplification technol. The expression profile of melanoma cells vs. normal cells was compared with that of conventional fluorescence detection. RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 25 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2000:240350 CAPLUS << LOGINID::20080906>>

DN 133:145756

TI mtDNA tandem repeats in domestic dogs and wolves: mutation mechanism studied by analysis of the sequence of imperfect repeats

AU Savolainen, Peter; Arvestad, Lars; Lundeberg, Joakim CS Department of Biotechnology, Royal Institute of Technology (KTH), Stockholm, S-100 44, Swed.

SO Molecular Biology and Evolution (2000), 17(4), 474-488 CODEN: MBEVEO: ISSN: 0737-4038

PB Society for Molecular Biology and Evolution

DT Journal

LA English

AB The ***mitochondrial*** (***mt***) ***DNA*** control region (CR) of dogs and wolves contains an *** array*** of imperfect 10 bp tandem repeats. This region was studied for 14 domestic dogs representing the four major phylogenetic groups of nonrepetitive CR and for 5 wolves. Three repeat types were found among these individuals, distributed so that different sequences of the repeat types were formed in different mols. This enabled a detailed study of the arrays and of the mutation events that they undergo. Extensive heteroplasmy was obsd. in all individuals; 85 different array types were found in one individual, and the total no. of types was estd. at 384. Among unrelated individuals, no identical mols. were found, indicating a high rate of evolution of the region. By performing a pedigree anal., array types which had been inherited from mother to offspring and array types which were the result of somatic mutations, resp., could be identified, showing that about 20% of the mols. within an individual had somatic mutations. By direct pairwise comparison of the mutated and the original array types, the physiognomy of the inserted or deleted elements (indels) and the approx. positions of the mutations could be detd. All mutations could be explained by replication slippage or point mutations. The majority of the indels were 1-5 repeats long, but deletions of up to 17 repeats were found. Mutations were found in all parts of the arrays, but at a higher frequency in the 5' end. Furthermore, the inherited array types within the motheroffspring pair were aligned and compared so that germ line mutations could be studied. The pattern of the germ line mutations was approx. the same as that of the somatic mutations.

RE ONT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 26 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2000:87041 CAPLUS < LOGINID::20080906>>

DN 133:15942

TI Chronic diarrhea associated with the A3243G mtDNA mutation

AU Santorelli, Filippo M.; Villanova, Marcello; Malandrini, Alessandro; Grieco, Gaetano S.; Palmeri, Silvia; Merlini, Luciano; Casali. Carlo

CS Neurological Institute, La Sapienza University, Rome, Italy

SO Neurology (2000), 54(1), 266-267 CODEN: NEURAI; ISSN: 0028-3878

PB Lippincott Williams & Wilkins

DT Journal

LA English

AB Diseases due mtDNA mutations are commonly referred to as "mitochondrial encephalomyopathies" because they preferentially affect tissues with high metabolic requirements such as brain and skeletal muscle. Clin. presentations are heterogeneous: diabetes mellitus, hearing loss, cardiomyopathy, or encephalomyopathy. This heterogeneity is particularly true for mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes syndrome (MELAS) assocd, with the A3243G mutation. We report an Italian woman with severe gastrointestinal involvement as the prominent presenting symptom of MELAS. Case history. A 33-yr-old woman had an 8-yr history of abdominal pain and watery diarrhea, with five to eight discharges per day, with progressive wt. loss and cachexia. Repeated biopsies of the large intestine excluded coeliac disease, Crohn's disease, and ulcerative colitis and showed only nonspecific atrophy of the intestinal villi. Anti-gliadin and anti-endomysial antibodies were absent. At 32 yr of age, she was referred because of tonic-clonic seizures. On neurol. examn., slight dysarthria, bilateral finger-to-nose dysmetria, gait ataxia, and bilateral hearing loss were found. Blood lactate was elevated. Bilateral calcification of the lenticular nuclei and cortical-subcortical atrophy and hypoplastic cerebellar vermis were seen by neuroimaging studies. Family history was unremarkable. A muscle biopsy of the left quadriceps did not show ragged-red fibers. A few cytochrome c oxidase (COX)deficient fibers were obsd. No other muscle analyses were performed. Ultrastructural examn. of a rectal biopsy disclosed aggregates of morphol. altered mitochondria and vacuolization in smooth muscle cells (figure), endothelial cells, and, to a lesser extent, enteric neurons. The A3243G mutation in the mitochondrial tRNALeu(UUR) gene was detected by sequence; restriction fragment length polymorphism analyses for quantitation of heteroplasmy used reported methodologies. The proportion of mutated genomes in the rectal biopsy (70%) was higher than in blood (53%). In the past 10 yr, a variety of human diseases have been assocd. with a heterogeneous ***array*** of ***mtDNA*** alterations. Gastrointestinal dysmotility, including diarrhea and pseudo-obstruction syndrome, is a cardinal feature of the syndrome of myoneurogastrointestinal encephalopathy (MNGIE), which is often assocd. with multiple mtDNA deletions. Abnormal-appearing mitochondria have been reported in smooth muscle cells and ganglion neurons in patients with MNGIE. Gastrointestinal manifestations are less frequent in patients with mtDNA point mutations such as MELAS. In addn., we obsd. a family harboring the A8344G mutation, in whom an individual underwent surgery for pseudo-obstruction (unpublished data, 1998). Although a striking protective effect against the phenotypical expression of the A3243G mutation was found in transformant cybrids harboring as little as 6% residual wild-type mtDNA, the precise threshold for abnormal function for different tissues, including the large intestine, is unknown. In our patient, 70% mutated mtDNA was found in the large intestine, likely reaching the crit. level for abnormal function. Our findings indicate that gastrointestinal symptoms can be an initial manifestation of the A3243G mutation. Whether they result from defective energy output in enteric neurons, smooth muscle or both, will require further research.

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 27 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2000:8287 CAPLUS << LOGINID::20080906>>

DN 132:304186

TI Mitochondrial DNA rearrangements in aging human brain and in situ PCR of mtDNA

AU Melova, S.; Schneider, J. A.; Coskun, P. E.; Bennett, D. A.; Wallace, D. C.

 $\ensuremath{\mathsf{CS}}$ Center For Molecular Medicine, Emory University, Atlanta, GA, USA

SO Neurobiology of Aging (1999), 20(5), 565-571 CODEN: NEAGDO; ISSN: 0197-4580

PB Elsevier Science Inc.

DT Journal

LA English

AB Deletions of the mitochondrial DNA (mtDNA) have been shown to accumulate with age in a variety of species regardless of mean or maximal life span. This implies that such mutations are either a mol. biomarker of senescence or that they are more causally linked to senescence itself. One assay that can be used to detect these mtDNA mutations is the long-extension polymerase chain reaction assay. This assay amplifies .apprx.16 kb of the mtDNA in mammalian mitochondria and preferentially amplifies mtDNAs that are either deleted or duplicated. We have applied this assay to the aging human brain and found a heterogeneous ***array*** of rearranged ***mtDNAs***. In addn., we have developed in situ polymerase chain reaction to detect mtDNA within individual cells of both the mouse and the human brain as a first step in identifying and enumerating cells contg. mutant mtDNAs in situ.

RE.ONT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 28 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1999:619953 CAPLUS << LOGI NI D::20080906>> DN 132:150164

TI Analysis of gene expression in multiple sclerosis lesions using cDNA microarrays

AU Whitney, Laurie Ward; Becker, Kevin G.; Tresser, Nancy J.; Caballero-Ramos, Carla I.; Munson, Peter J.; Prabhu, Vinayakumar V.; Trent, Jeffrey M.; McFarland, Henry F.; Biddison, William E.

CS Molecular Immunology Section, Neuroimmunology Branch, National Institute, National Institutes of Health, Bethesda, MD, 20892-1400, USA

SO Annals of Neurology (1999), 46(3), 425-428 CODEN: ANNED3; ISSN: 0364-5134

PB Lippincott Williams & Wilkins

DT Journal

LA English

AB In multiple sclerosis (MS) patients, a coordinated attack of the immune system against the primary constituents of oligodendrocytes and/or the myelin sheath of oligodendrocytes results in the formation of lesions in the brain and spinal cord. Thus far, however, a limited no. of genes that potentially contribute to lesion pathol. have been identified. Using cDNA microarray technol., the authors have performed expts. on MS tissue monitoring the expression pattern of over 5,000 genes and compared the gene expression profile of normal white matter with that found in acute lesions from the brain of a single MS patient. Sixty-two differentially expressed genes were identified, including the Duffy chemokine receptor, interferon regulatory factor-2, and tumor necrosis factor alpha receptor-2 among others. Thus, cDNA microarray technol, represents a powerful new tool for the identification of genes not previously assocd. with the MS disease process.

RE.ONT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 29 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1999:530238 CAPLUS << LOGINI D:: 20080906>>

DN 131:332782

TI A Novel Mitochondrial DNA-like Sequence in the Human Nuclear Genome

AU Herrnstadt, Corinna; Gevenger, William; Ghosh, Soumitra S.; Anderson, Christen; Fahy, Eoin; Miller, Scott; Howell, Neil; Davis. Robert E.

CS MitoKor, San Diego, CA, 92121, USA

SO Genomics (1999), 60(1), 67-77 CODEN: GNMCEP; ISSN: 0888-7543

PB Academic Press

DT Journal

LA English

AB The authors describe here a nuclear mitochondrial DNA-like sequence (numtDNA) that is nearly identical in sequence to a continuous 5842 bp segment of human mitochondrial DNA (mtDNA) that spans nucleotide positions 3914 to 9755. On the basis of evolutionary divergence among modern primates, this numtDNA mol. appears to represent mtDNA from a hominid ancestor that has been translocated to the nuclear genome during the recent evolution of humans. This numtDNA sequence harbors synonymous and nonsynonymous nucleotide substitutions relative to the authentic human *** mtDNA*** sequence, including an ***array*** of substitutions that was previously found in the cytochrome c oxidase subunit 1 and 2 genes. These substitutions were previously reported to occur in human mtDNA, but subsequently contended to be present in a nuclear pseudogene sequence. The authors now demonstrate their exclusive assocn. with this 5842-bp numtDNA, which the authors have characterized in its entirety. This numtDNA does not appear to be expressed as a mtDNA-encoded mRNA. It is present in nuclear DNA from human blood donors, in human SH-SY5Y and A431 cell lines, and in .rho.0 SH-SY5Y and .rho.0 A431 cell lines that were depleted of mtDNA. The existence of human numtDNA sequences with great similarities to human mtDNA renders the amplification of pure mtDNA from cellular DNA very difficult, thereby creating the potential for confounding studies of mitochondrial diseases and population genetics. (c) 1999 Academic Press.

RE.ONT 36 THERE ARE 36 CLTED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 30 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1998:192961 CAPLUS < LOGINID::20080906>>

DN 128:306719

OREF 128:60753a,60756a

TI Mutations in mitochondrial DNA accumulate differentially in three different human tissues during aging

AU Liu, Vincent W. S.; Zhang, Chunfang; Nagley, Phillip

CS Department of Biochemistry and Molecular Biology, Monash University, Clayton, 3168, Australia

SO Nucleic Acids Research (1998), 26(5), 1268-1275 CODEN: NARHAD; ISSN: 0305-1048

PB Oxford University Press

DT Journal

LA English

AB In 60 human tissue-samples (encompassing skeletal muscle, heart and kidney) obtained from subjects aged from under 1 to 90 yr, the authors used quant. PCR procedures to quantify mitochondrial DNA (mtDNA) mols. carrying the 4977 bp deletion

(mtDNA4977) and 3243 A.fwdarw.G base substitution. In addn... the prevalence of multiple mtDNA deletions was assessed in a semi-quant. manner. For all three tissues, the correlations between the accumulation of the particular mtDNA mutations and age of the subject are highly significant. However, differential extents of accumulation of the two specific mutations in the various tissues were obsd. Thus, the mean abundance (percentage of mutant mtDNA out of total mtDNA) of mtDNA4977 in a subset of age-matched adults is substantially higher in skeletal muscle than in heart and kidney. However, the mean abundance of the 3243 A.fwdarw. G mutation in skeletal muscle was found to be lower than that in heart and kidney. Visualization of *** arrays*** of PCR products arising from multiple *** mtDNA*** deletions in DNA extd. from adult skeletal muscle, was readily made after 30 cycles of PCR. By contrast, in DNA extd. from adult heart or kidney, amplification for 35 cycles of PCR was required to detect multiple mtDNA deletions. Although such multiple deletions are less abundant in heart and kidney than in skeletal muscle, in all tissue exts. there are unique patterns of bands, even from different tissues of the same subject. The differential accumulation of mtDNA4977. other mtDNA deletions and the 3243 A.fwdarw.G mutation in the three tissues analyzed presumably reflects different metabolic and senescence characteristics of these various tissues. RE. CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE **FORMAT**

L20 ANSWER 31 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1998:144425 CAPLUS < LOGINI D::20080906>> DN 128:253627

OREF 128:50115a,50118a

TI Tandem repeat polymorphism and heteroplasmy in the mitochondrial control region of redfishes (Sebastes: Scorpaenidae)

AU Bentzen, P.; Wright, J. M.; Bryden, L. T.; Sargent, M.; Zwanenburg, K. C. T.

CS Mar. Biotechnol. Lab., Univ. Washington, Seattle, WA, 98105, USA

SO Journal of Heredity (1998), 89(1), 1-7 CODEN: JOHEA8; ISSN: 0022-1503

PB Oxford University Press

DT Journal

LA English

AB Three species of redfish (Sebastes) share a common pattern of mitochondrial DNA tandem repeat polymorphism and heteroplasmy in the northwest Atlantic Ocean. All three species exhibit 9-17 copies of an approx. 275 base pair (bp) tandem repeat situated within the 3' domain of the control region. Sequence anal. of cloned ***mtDNA*** from S. mentella revealed that the tandem ***array*** is adjacent to the tRNAphe gene, and that the repeat shares 53% identity with the tRNAphe, gene and part of the 12S rRNA gene. These features, as well as potential secondary structure assumed by the repeat, are consistent with previously proposed models explaining tandem duplications in the 3' end of the control region. In a sample comprising 36 S. fasciatus, 52 S. mentella, and 13 S. marinus taken near Newfoundland, neither the mean no. of repeats per fish (12.2-12.7) nor the frequency of heteroplasmy varied significantly among species. A total of 42% of the redfishes were heteroplasmic, bearing either two or three repeat variants (33% and 9%, resp.). The similarity of the frequency distributions of tandem repeat variants in the three species suggests either a common balance between mutation and selection in the three species, or mitochondrial gene flow between them.

RE.ONT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 32 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1997:330250 CAPLUS << LOGINID::20080906>>

DN 127:45927

OREF 127:8655a

TI Animal mitochondrial DNA recombination

AU Lunt, David H.; Hyman, Bradley C.

CS Dep. Biology, Univ. California, Riverside, CA, 92521, USA

SO Nature (London) (1997), 387(6630), 247 CODEN: NATUAS; ISSN: 0028-0836

PB Macmillan Magazines

DT Journal

LA English

AB Contrary to the consensus view that genetic recombination does not occur in animal mitochondrial DNA (mtDNA), the authors found end-products of recombination in the mitochondrial genome of the phytonematode, Meloidogyne javanica. Sequences of nematode ***mtDNA*** VNTRs frequently contained deletions in the center of the ***array***, indicating that genetic recombination might be occurring. Small circular mols. composed of M. javanica mtDNA sequences were detected using PCR and subsequent sequence anal.; these appear to be subgenomic recombination end-products. RE.CNT 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE REFORMAT

L20 ANSWER 33 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1996:156010 CAPLUS << LOGINI D:: 20080906>>

DN 124:252132

OREF 124:46505a.46508a

TI Length variation, heteroplasmy and sequence divergence in the mitochondrial DNA of four species of sturgeon (Acipenser) AU Brown, James R.; Beckenbach, Karen; Beckenbach, Andrew

T.; Smith, Michael J.

CS Evolutionary Biology Program, Canadian Institute for Advanced Research, Halifax, NS, B3H 4H7, Can.

SO Genetics (1996), 142(2), 525-35 CODEN: GENTAE; ISSN: 0016-6731

PB Genetics Society of America

DT Journal

LA English

AB The extent of mtDNA length variation and heteroplasmy as well as DNA sequences of the control region and two tRNA genes were detd. for four North American sturgeon species: Acipenser transmontanus, A. medirostris, A. fulvescens and A. oxyrhynchus. Across the Continental Divide, a division in the occurrence of length variation and heteroplasmy was obsd. that was concordant with species biogeog. as well as with phylogenies inferred from restriction fragment length polymorphisms (RFLP) of whole mtDNA and pairwise comparisons of unique sequences of the control region. In all species, ***mtDNA*** length variation was due to repeated ***arrays*** of 78-82-bp sequences each contg. a D-loop strand synthesis termination assocd. sequence (TAS). Individual repeats showed greater sequence conservation within individuals and species rather than between species, which is suggestive of concerted evolution. Differences in the frequencies of multiple copy genomes and heteroplasmy among the four species may be ascribed to differences in the rates of recurrent mutation. A mechanism that may offset the high rate of mutation for increased copy no. is suggested on the basis that an increase in the no. of functional TAS motifs might

reduce the frequency of successfully initiated H-strand replications.

L20 ANSWER 34 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1994:596939 CAPLUS << LOGINI D::20080906>>

DN 121:196939

OREF 121:35587a,35590a

TI Mitochondrial DNA sequence variation in human evolution and disease

AU Wallace, Douglas C.

CS Department of Genetics and Molecular Medicine, Emory University School of Medicine, Atlanta, GA, 30322, USA

SO Proceedings of the National Academy of Sciences of the United States of America (1994), 91(19), 8739-46 CODEN: PNASA6; ISSN: 0027-8424

DT Journal; General Review

LA English

AB A review with 101 refs. Germ-line and somatic mtDNA mutations are hypothesized to act together to shape our history and our health. Germ-line mtDNA mutations, both ancient and recent, have been assocd. with a variety of degenerative diseases. Mildly to moderately deleterious germ-line mutations, like neutral polymorphisms, have become established in the distant past through genetic drift but now may predispose certain individuals to late-onset degenerative diseases. As an example, a homoplastic, Caucasian, tRNAGIn mutation at nucleotide pair (np) 4336 has been obsd. in 5% of Alzheimer disease and Parkinson disease patients and may contribute to the multifactorial etiol. of these diseases. Moderately to severely deleterious germ-line mutations, on the other hand, appear repeatedly but are eliminated by selection. Hence, all extant mutations of this class are recent and assocd. with more devastating diseases of young adults and children. Representative of these mutations is a heteroplasmic mutation in MTND6 at np 14459 whose clin. presentations range from adult-onset blindness to pediatric dystonia and basal ganglial degeneration. To the inherited mutations are added somatic *** mtDNA*** mutations which accumulate in random *** arrays*** within stable tissues. These mutations provide a mol. clock that measures our age and may cause a progressive decline in tissue energy output that could ppt. the onset of degenerative diseases in individuals harboring inherited deleterious mutations.

L20 ANSWER 35 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1994:526412 CAPLUS << LOGINID::20080906>>

DN 121:126412

OREF 121:22645a,22648a

TI Mapping and repeated sequence organization of mitochondrial DNA in scallop, Pecten maximus

AU Rigaa, Ali; Le Gal, Yves; Sellos, Daniel; Monnerot, Monique

CS Lab. Biol. Mar., Coll. France, Concarneau, 29900, Fr.

SO Molecular Marine Biology and Biotechnology (1993), 2(4), 218-24 CODEN: MMBBEQ; ISSN: 1053-6426

DT Journal

LA English

AB Mitochondrial DNA (mt DNA) of scallop, Pecten maximus, analyzed from 3 populations from the western coast of France, exhibits an important size polymorphism with respect to restriction fragment patterns. From 27 individuals studied, 6 size classes of mitochondrial genomes are obsd. within a range of 20 to 25.8 kb. The restriction map of the ***mtDNA*** of Pecten maximus shows that this genome contains a tandem ***array*** of 1.6 kb repeat units. Variation among mtDNA mols. in the copy no. of this repeat unit (2-5 copies) results in mtDNA length variation and size heteroplasmy. The digestion patterns for all restriction endonucleases used and the mapping

of the restriction sites indicate that an event of addn./deletion of approx. 500 to 1,000 bp has occurred in one of the repeated units for 2 mtDNA samples.

L20 ANSWER 36 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1991:179036 CAPLUS << LOGINID::20080906>> DN 114:179036

OREF 114:30055a,30058a

TI Mitochondrial DNA mutations associated with neuromuscular diseases: analysis and diagnosis using the polymerase chain reaction

AU Wallace, Douglas C.; Lott, Marie T.; Lezza, Angela M. S.; Seibel, Peter; Voljavec, Alexander S.; Shoffner, John M. CS Sch. Med., Emory Univ., Atlanta, GA, 30322, USA

SO Pediatric Research (1990), 28(5), 525-8 CODEN: PEREBL; ISSN: 0031-3998

DT Journal; General Review

LA English

AB A rev., with 32 refs. A no. of neuromuscular diseases are assocd. with mol. defects in the mitochondrial DNA (mtDNA). These include: 1) a missense mutation at nucleotide 11778 in the *** mtDNA*** of Leber's hereditary optic neuropathy patients; 2) a heterogeneous ***array*** of deletions in the *** mtDNA*** of ocular myopathy patients; and 3) small deletions and point mutations in the mtDNA of myoclonic epilepsy and ragged red fiber disease patients. These diseases can now be diagnosed at the mol. level from small patient samples by amplifying the affected mtDNA regions using the polymerase chain reaction. Leber's hereditary optic neuropathy is diagnosed through loss of an SfaNI restriction site. Ocular myopathy deletions are identified by differential amplification across deletion breakpoints. Familial diseases such as myoclonic epilepsy and ragged red fiber disease might be diagnosed by identifying small deletions through amplification and electrophoretic anal. of the entire mtDNA genome or by identifying point mutations through differential oligonucleotide hybridization. As addnl. mtDNA mol. defects are identified, mol. anal. will likely become a primary tool for the diagnosis of these diseases.

L20 ANSWER 37 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1990:136267 CAPLUS << LOGINID::20080906>>

DN 112:136267

OREF 112:22977a,22980a

TI A genetic discontinuity in a continuously distributed species: mitochondrial DNA in the American oyster, Crassostrea virginica AU Reeb, Carol A.; Avise, John C.

CS Dep. Genet., Univ. Georgia, Athens, GA, 30602, USA SO Genetics (1990), 124(2), 397-406 CODEN: GENTAE; ISSN: 0016-6731

DT Journal

LA English

AB Restriction site variation in mitochondrial DNA (mtDNA) of the American oyster (C. virginica) was surveyed in continuously distributed populations sampled from the Gulf of St. Lawrence, Canada, to Brownsville, Texas. The mtDNA clonal diversity was high, with 82 different haplotypes revealed among 212 oysters with 13 endonucleases. The *** mtDNA*** clones grouped into 2 distinct genetic *** arrays*** (estd. to differ by .apprx.2.6% in nucleotide sequence) that characterized oysters collected north vs. south of a region on the Atlantic mid-coast of Florida. The population genetic break in mtDNA contrasts with previous reports of near uniformity of nuclear (allozyme) allele frequencies throughout the range of the species, but agrees closely with the magnitude and pattern of mtDNA differentiation reported in other estuarine species in the southeastern United

States. This concordance of mtDNA phylogenetic pattern across independently evolving species provides strong evidence for vicariant biogeog. processes in initiating intraspecific population structure. The post-Miocene ecol, history of the region suggests that reduced pptn. levels in an enlarged Floridian peninsula may have created discontinuities in suitable estuarine habitat for oysters during glacial periods, and that today such population sepns. are maintained by the combined influence of ecol. gradients and oceanic currents on larval dispersal. The results are consistent with the hypothesis that historical vicariant events, in conjunction with contemporary environmental influences on gene flow, can result in genetic discontinuities in continuously distributed species with high dispersal capability.

L20 ANSWER 38 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1989:207107 CAPLUS << LOGINID::20080906>> DN 110:207107

OREF 110:34259a,34262a

TI Stable maintenance of a 35-base-pair yeast mitochondrial

AU Fangman, Walton L.; Henly, John W.; Churchill, Gary; Brewer, Bonita J.

CS Dep. Genet., Univ. Washington, Seattle, WA, 98195, USA SO Molecular and Cellular Biology (1989), 9(5), 1917-21 CODEN: MCEBD4; ISSN: 0270-7306

DT Journal

LA Enalish

AB Small deletion variants ([rho-] mutants) derived from the wild-type ([rho+]) Saccharomyces cerevisiae mitochondrial genome were isolated and characterized. The mutant mitochondrial DNAs (mtDNAs) examd. retained as little as 35 base pairs of one section of intergenic DNA, were composed entirely of A.cntdot.T base pairs, and were stably maintained. These simple *** mtDNAs*** existed in tandemly repeated * * * arravs * * * at an amplified level that made up .apprx.15% of the total cellular DNA and, as judged by fluorescence microscopy, had a nearly normal mitochondrial arrangement throughout the cell cytoplasm. The simple nature of these [rho-] genomes indicates that the sequences required to maintain mtDNA must be extremely simple.

L20 ANSWER 39 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1988:606080 CAPLUS << LOGINI D::20080906>>

DN 109:206080

OREF 109:33979a,33982a

TI Telomeric repeats of Tetrahymena malaccensis mitochondrial DNA: a multimodal distribution that fluctuates erratically during growth

AU Morin, Gregg B.; Cech, Thomas R.

CS Dep. Chem. Biochem., Univ. Colorado, Boulder, CO, 80309-

SO Molecular and Cellular Biology (1988), 8(10), 4450-8

CODEN: MCEBD4; ISSN: 0270-7306

DT Journal

LA English

AB The linear mitochondrial DNA (mtDNA) of T. malaccensis has tandem 52-base-pair repeats at its telomeres. The mtDNA has a multimodal distribution of telomeres. Different groups in the distribution have different nos. of telomeric repeats. The std. deviation of the size of each end group is independent of the mean size of the end group. The 2 sides of the mtDNA have different multimodal distributions of repeats. Goned cell lines have multimodal distributions of mtDNA telomeres distinct from that of the original cell line. The no. of telomere end groups and the av. size of the end groups change in an erratic fashion as the cells are passaged and do not reach a stable equil. distribution in

185 generations. It is proposed that the mean size of a telomere end group and the size distribution of an end group are independently regulated. The system controlling the av. size of end groups may be defective in T. malaccensis, since a closely related species (T. thermophila) does not have a multimodal distribution of mtDNA telomeres. T. hyperangularis, which has different telomeric repeats on each side of its mtDNA, has a multimodal distribution of mtDNA telomeres on only one side, suggesting that the mechanism controlling the av. no. of repeats in an end group can be sequence specific. These mitochondrial telomeres provide a new example of the more general phenomenon of expansion and contraction of ***arrays*** of repeated sequences seen, for example, with simple-sequence satellite ***DNAs***; however, the *** mitochondrial*** telomeres change on a very short time scale.

L20 ANSWER 40 OF 40 CAPLUS COPYRIGHT 2008 ACS on STN AN 1988:162512 CAPLUS << LOGINI D:: 20080906>> DN 108:162512

OREF 108:26591a,26594a

TI Phylogeographic population structure of red-winged blackbirds assessed by mitochondrial DNA

AU Ball, R. Martin, Jr.; Freeman, Scott; James, Frances C.; Bermingham, Eldredge; Avise, John C.

CS Dep. Genet., Univ. Georgia, Athens, GA, 30602, USA SO Proceedings of the National Academy of Sciences of the United States of America (1988), 85(5), 1558-62 CODEN: PNASA6; ISSN: 0027-8424

DT Journal LA English

AB A continent-wide survey of restriction-site variation in mitochondrial DNA (mtDNA) of the Red-winged Blackbird (Agelaius phoeniceus) was conducted to assess the magnitude of phylogeog, population structure in an avian species. A total of 34 mtDNA genotypes was obsd. among the 127 specimens assayed by 18 restriction endonucleases. Nonetheless, population differentiation was minor, as indicated by small genetic distances in terms of base substitutions per nucleotide site between mtDNA genotypes (max.) and by the widespread geog. distributions of particular *** mtDNA*** clones and phylogenetic **arrays*** of clones. Extensive morphol. differentiation among redwing populations apparently has occurred in the context of relatively little phylogenetic sepn. A comparison between mtDNA data sets for Red-winged Blackbirds and deermice (Peromyscus maniculatus) also sampled from across North America shows that intraspecific population structures of these 2 species differ dramatically. The lower phylogeog. differentiation in redwings is probably due to historically higher levels of gene flow.

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| | L11 | 260 S L10 NOT | 1989/PY | | | | |
|---------------------|----------------------|----------------|----------|------------|---------------|----|--|
| | L12 | 257 S L11 NOT | 1988/PY | | | | |
| | L13 | 249 S L12 NOT | PY< 1990 | | | | |
| | L14 | 305 S ((((MITO | CHONDRI? | OR MT)(5A) | (DNA# (| OR | |
| | CDNA# C | OR GENE#)) OR | MTDNA | | | | |
| | L15 | 276 S L14 NOT | 2008/PY | | | | |
| | L16 | 215 S L15 NOT | 2007/PY | | | | |
| | L17 | 165 S L16 NOT | 2006/PY | | | | |
| | L18 | 171 S L16 NOT | 2005/PY | | | | |
| | L19 | 139 S L18 NOT | 2004/PY | | | | |
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